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# Early Onset of Levodopa-Induced Dyskinesia in Parkinson's Diseased Patient: A Case Report

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**ABSTRACT:** A 70-year-old woman with a prior cerebrovascular accident presented with classical Parkinsonian features and an atypical ulnar deviation of the wrist. A robust response to levodopa/carbidopa confirmed idiopathic Parkinson's disease, but she developed severe levodopa-induced dyskinesia within two days of therapy initiation. Laboratory tests showed leucocytosis and markedly elevated ESR, while negative rheumatoid factor testing excluded rheumatological causes for the striatal hand deformity, supporting its attribution to Parkinson's disease–associated dystonia. Discontinuation of levodopa led to complete resolution of the dyskinesia. This case highlights the diagnostic challenges of Parkinson's disease in patients with comorbidities, the clinical relevance of striatal hand deformity, and the need for cautious titration of levodopa to minimize early-onset dyskinesia.

**KEYWORDS:** Parkinson's disease, Levodopa, Dyskinesia, Striatal hand deformity, Case report, Adverse drug reaction.

## I. INTRODUCTION

Parkinson's disease (PD) is the second most common neurodegenerative disorder, primarily characterized by the cardinal motor symptoms of resting tremor, bradykinesia, rigidity, and postural instability. While the diagnosis is predominantly clinical, atypical presentations can pose significant diagnostic challenges, particularly in patients with complex medical histories [1]. The therapeutic response to levodopa, a dopamine precursor, is a key diagnostic criterion, but its use carries the risk of inducing adverse motor complications, particularly dyskinesias [2].

This report details a case of a patient with a history of cerebrovascular accident (CVA) presenting with classic Parkinsonian symptoms alongside an atypical feature, ulnar deviation of the wrist. The patient's subsequent rapid development of levodopa-induced dyskinesia not only confirmed idiopathic PD but also highlighted the complex relationship between disease, motor manifestations, and pharmacological treatment [3].

## II. CASE SUMMARY

A 70-year-old female presented with the complaint of seizure (single episode), right knee pain, a 6-month history of involuntary rhythmic tremors, stiffness and decreased dexterity in the right upper limb, reduced locomotor movements, and self-directed speech. Written Informed consent was obtained from the patient legal guardian for publication of this case report.The patient was initiated on a standard regimen of levodopa/carbidopa 110mg TDS. Following the first day of treatment, her tremor and rigidity showed a remarkable improvement. However, within just 2 days of therapy initiation, she began to experience non-rhythmic involuntary movements of the upper limbs and face. This drug-induced dyskinesia, despite a positive therapeutic response to levodopa, confirmed the underlying diagnosis of idiopathic Parkinson's disease.

## III. PAST HISTORY

The patient had a known history of old CVA with left-sided hemiparesis, largely recovered, for which she was on antiplatelet and statin therapy (Aspirin 150 mg BD and Atorvastatin 10 mg). She also had urosepsis with right pyelonephritis, seizure disorder (on Phenytoin 100 mg), and a past episode of septic encephalopathy (recovered).

## IV. VITAL SIGNS AND PHYSICAL EXAMINATION

Examination revealed stable hemodynamics: temperature 37.2°C, blood pressure 120/80 mmHg, heart rate 83/min, respiratory rate 18/min, and oxygen saturation 99% on room air. Cardiovascular and respiratory examinations were unremarkable. On examination showed tremor and rigidity in the wrist and joints. A pronounced resting tremor was observed in both hands and arms, with significant rigidity upon passive movement of the wrist and elbow. An unusual finding was mild but persistent ulnar deviation of the right wrist.

Sensation and reflexes were intact. The patient's prior left-sided weakness from CVA was distinct from the new-onset motor symptoms. Based on tremor, rigidity, and bradykinesia, idiopathic Parkinsonism was clinically diagnosed.

## V. LABORATORY INVESTIGATIONS

The total white blood cell count was marginally elevated at 17.8 × 103 cells/µL. Haemoglobin was 11 g/dl and platelet count 396 × 103/µL remained within normal parameters. Biochemical analysis of liver function tests, ALT at 21 U/L and AST at 26 U/L, were within normal range. Renal function tests including serum creatinine was 0.67 mg/dL and urea was 24 mg/dL, which were within normal limits. ESR was >140 mm, CRP was 4.2 mg/L, and rheumatoid factor was negative.

## VI. COURSE IN THE HOSPITAL

During subsequent ward rounds, the patient developed significant levodopa-induced dyskinesia. Considering the severity, levodopa was withdrawn. Post-withdrawal, dyskinesia resolved, confirming its drug-induced nature, though the original Parkinsonian motor symptoms returned.

The adverse drug reaction (ADR) was reported to the Adverse Drug Monitoring Centre, Government Medical College, Nagapattinam. Causality assessment using the Naranjo scale classified the reaction as probable.

## VII. DISCUSSION

This case illustrates the diagnostic complexity of idiopathic Parkinson's disease (PD), which often manifests with both motor and non-motor features that may be obscured by coexisting medical conditions. The patient presented with a typical Parkinsonian tremor alongside the non-motor feature of "self-talk," consistent with the broad spectrum of symptoms seen in idiopathic PD that affect motor and cognitive domains [1]. An important clinical distinction was recognizing the patient's history of self-directed speech as separate from hallucinations, the latter being a recognized complication of dopaminergic treatment and a crucial factor in therapeutic decision-making. Although the prior history of cerebrovascular accident (CVA) raised the possibility of vascular parkinsonism, the presence of classical motor signs and a robust levodopa response argued against this alternative diagnosis [2,3]. The presence of a "striatal hand deformity" further reinforced idiopathic PD, as this dystonic feature is characteristic of the disease and not typical of rheumatological disorders such as rheumatoid arthritis [4,5].

Laboratory investigations showed a markedly elevated white blood cell count (17.8 × 109/L), an erythrocyte sedimentation rate >140 mm/hr, and increased C-reactive protein (4.2 mg/L), suggesting a significant inflammatory process. However, the absence of rheumatoid factor was critical in excluding a rheumatological etiology for the hand deformity. This finding supported the conclusion that the deformity was related to PD-associated dystonia rather than inflammatory arthritis.

From a pharmacological standpoint, levodopa's therapeutic efficacy lies in its ability to cross the blood–brain barrier and undergo enzymatic conversion to dopamine via L-aromatic amino acid decarboxylase [6]. The patient's substantial symptomatic improvement following levodopa initiation confirmed a dopaminergic deficit, consistent with idiopathic PD. Nevertheless, the emergence of levodopa-induced dyskinesia underscored the narrow therapeutic window and individual variability in drug response.

A further complexity in management was the patient's polypharmacy, which included aspirin, atorvastatin, enalapril, and carbamazepine. While levodopa is generally compatible with common cardiovascular medications, the potential interaction with carbamazepine—an anticonvulsant—was clinically relevant, as such drugs may alter levodopa's pharmacokinetics and reduce its efficacy [7]. Careful monitoring of patients on this combination is essential to preserve treatment effectiveness.

A key consideration was the safety of prescribing levodopa to an individual with a seizure history. Evidence indicates that levodopa does not significantly lower the seizure threshold and is generally safe in patients with well-controlled epilepsy [8]. In this patient, the benefits of levodopa in relieving disabling motor symptoms outweighed the minimal risk of seizure exacerbation. Importantly, the patient's marked improvement after levodopa initiation became the strongest diagnostic confirmation. However, the subsequent development of early-onset dyskinesia and progression to hallucinations represented clinically significant adverse drug reactions (ADRs). Dyskinesia is a well-documented long-term complication due to chronic dopaminergic stimulation [9], but its unusually rapid appearance in this case suggests heightened individual drug sensitivity [10].

## VIII. CONCLUSION

This case emphasizes the value of thorough clinical evaluation when differentiating idiopathic PD from secondary parkinsonism, particularly in patients with prior cerebrovascular disease. The coexistence of classic motor symptoms—tremor and striatal hand deformity—together with a positive levodopa response, strongly indicated idiopathic PD. Although the patient's concurrent medications included carbamazepine, which carries the potential for interaction, this did not preclude effective levodopa therapy. Moreover, the case demonstrates that levodopa can be safely used in patients with a stable seizure history. The patient's rapid onset of levodopa-induced dyskinesia highlighted the importance of individualized dosing strategies and close monitoring for adverse effects. This aligns with the "start low, go slow" principle, which remains essential in minimizing complications and optimizing therapeutic outcomes [11]. Future investigations may help identify genetic or pathological markers that predispose certain patients to early complications of dopaminergic therapy, thereby guiding more personalized treatment approaches.

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